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ANSWER I OF 25 MEDLINE on STN
the thalausus in RHSPS was observed at 1, 2 and 4 weeks after
distal MCAO. In addition, intracerebroventricular infusion of
NEP1-40, a Nogo-6 receptor (NgR) antagonist peptide,
was administered starting 24 h after MCAO and continued for 1, 2 and 4.
the expression of Nogo-A in oligodendrocytes increased persistently and
its localization became redistributed around damaged axons and dendrites.
Administration of NgP2-40 downregulated the expression
of Nogo-A, reduced axonal injury and enhanced axonal regeneration. Our
data suggest that Nogo-A is involved in L14 AB

^{=&}gt; s Lingo-1(w)antagonist L10 10 LINGO-1(W) ANTAGONIST

^{=&}gt; s li0 and (li or l2) Ll1 0 Ll0 AND (Ll OR L2)

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ANSWER 2 OF 25 MEDLINE on STN
inhibitors (MAI). To overcome CSPG- or myelin-induced
inhibitors (MAI). To overcome CSPG- or myelin-induced
inhibition, strategies based on extrinsic and intrinsic treatments have
been developed. For example, NEP1-40 is a synthetic
peptide that promotes axonal regeneration by blocking Nogo-66/NgR
interaction and chondroltinase ABC (ChABC), which degrades CS, thereby.
model, overexpressed CSPG and MAI impaired axon regrowth, which mimics
regeneration failure in vivo. Both CS cleavage with ChABC and
NEP1-40 strongly facilitated the regrowth of entorhinal
axons after axotomy, permitting the re-establishment of synaptic contacts
with target cells. However, the combined treatment did not improve the
regeneration induced by ChABC alone, and the delayed treatment of ChABC,
but not NEP1-40, had a less pronounced effect on
axonal regrowth compared with acute treatment. These results provide

insight into the development of.

0 (Myelin Protein); 0 (MRPL-40 protein, human); 0
(Mogo protein); 0 (Peptide Fragments); 0 (Proteochondroitin Sulfates); 0
(Receptors, Cell Surface); 0 (Rtn4r protein, mouse); EC

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ATT AB

and the objective genes were.

proteins were purified by Ni-column and detected by using Western-blot test. RESULTS: The Nogo-66 and NRP1-40 genes were successfully cloned from rat, which were 215 bp and 137 bp for each one when add the enzyme.

the results of electrophoresis. The protein nogo-66 and NRP1-40, which relative molecular weight were 31.2 1013) and 30.3 x 10(3) respectively. The results of Western-blot test confirmed that the antigenicity of the two proteins was precise. CONCLUSION: Nogo-66 and NRP1-40 proteins can be expressed in a high efficiency in vitro using genetic engineering, so it provides a good basis for. ANSWER 3 OF 25 MEDLINE on STN
Cloning of MRF1-40 gene and expression of its protein.

OBJECTIVE: To clone the genes of nogo-66 and NRF1-40
from spinal cord of rat and to realize the expression of its protein in vitro. MRTHODS: The nogo-66 and NRF1-40 genes were cloned from the spinal cord of juvenil rat by use of RT-PCR techniques.

ANSWER 4 OF 25 MEDLINE on STN
IN.1, the monoclonal antibody against the exon 3-encoded N-terminal domain
of Nogo-A, and the Nogo-6 receptor (NGR) antagonist NRP140 have both shown efficacy in promoting regeneration in animal
40 have both shown efficacy in promoting regeneration in animal
spinal cord injury models, the latter even when administered
subcutaneously 1. targeted disruption of Nogo and NgR have,
surprisingly, only modest regenerative capacity (if any) compared with
treatment with IN-1 or NEP1-40. Disruption of the
Nogo gene by various groups yielded results ranging from significant
regenerative improvement in young mice to number . background, we
suggest here some possible and testable explanations for the above
phenomena. These possiblilities include effects of IN-1 and NEP1
-40 on the CNS beyond neutralization of Nogo and NgR functions,
and the latter's possible role in the CNS beyond that. L14 AB

MEDLINE on STN ANSWER 5 OF 25 ₽ [7

therapeutic time window. Subcutaneous treatment with the NgR antagonist peptide Argard (Nggo extracellular peptide, residues 1-40) results in extensive growth of corticospinal axons, sprouting of serotonergic fibers, upregulation of axonal growth. protein 1A), and synapse re-formation. Locomotor recovery after thoracic spinal coinjury is enhanced. Furthermore, delaying the initiation of systemic NRB1-40 administration for up to 1 week after cord lessions does not limit the degree of axon sprouting and functional

recovery..

MEDLINE on STN ANSWER 6 OF 25

mid-thoracic spinal cord hemisection results in significant axon growth of the corticospinal tract, and improves functional recovery. Thus, Nogo-66 and NgR have central roles in limiting axonal regeneration after CNS injury, and NEP1-40 provides a potential therapeutic NgR. Here, we identify competitive antagonists of NgR derived from amino-terminal peptide fragments of Nogo-66. The Nogo-66(1 40) amaganist peptide (NRP1 40) blocks Nogo-66 or CNS myelin inhibition of axonal qutgrowth in vitro, demonstrating that NgR mediates a significant portion of axonal outgrowth inhibition by myelin. Intrathecal administration of NRP1 40 to rats with L14 AB

ANSWER 7 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN inhibitors (MAI). To overcome CSPC- or myelin-induced inhibition, strategies based on extrinsic and intrinsic treatments have been developed. For example, NEPL-40 is a synthetic developed. For example, NEPL-40 is a synthetic peptide that promotes aconal regeneration by blocking Nogo-66/NgR interaction and chondroitinase ABC (ChABC), which degrades C5, thereby. Tegeneration failure in vivo. Both CS cleavage with ChABC and NEPL-40 strongly facilitated the regrowth of entorhinal axons after axotomy, permitting the re-establishment of synaptic contacts with target cells. However, the combined treatment did not improve the regeneration induced by ChABC alone, and the delayed treatment of ChABC, but not NEPL-40, had a less pronounced effect on axonal regrowth compared with acute treatment. These results provide L14 AB.

insight into the development of.

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chondroitin sulfate proteoglycans, chondroitinase ABC [EC 4.2.2.4], myelin-associated glycoprotein, Nogo-66 receptor; oligodendrocyte myelin glycoprotein; MEP1-40

ANSWER 8 OF 25 BIOSIS COPPRIGHT (c) 2007 The Thomson Corporation on STN IN-1, the monoclonal antibody against the exon 3-encoded N-terminal domain of Nogo-A, and the Nogo-6 receptor (NgP) antagonist NEP1-440 have both shown efficacy in promoting regeneration in animal spinal cord injury models, the latter even when administered š subcutaneously.

subcutaneously.

surprisingly, only modest regenerative capacity (if any) compared wit treatment with IN-1 or NRP1-40. Disruption of the Nogo gene by various groups yielded results ranging from significant regenerative improvement in young mice to number.

Suggest here some possible and testable explanations for the above phenomena. These possibilities include effects of IN-1 and NRP1-40 on the CNS beyond neutralization of Nogo and NGR functions, and the latter's possible role in the CNS beyond that. R 17

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Spinal Cord Injuries (MeSH) Chemicals & Biochemicals system disease, injury

Nogo receptor; Nogo-66 receptor; neutralizing agent; IN-1; Nogo-A: N-terminal domain; NEP1-40: immunologic-drug, immunostimulant-drug, subcutaneous administration

two NgR. of CNS myelin than those that target specific myelin proteins the anti-NogoA antibody, IN-1 or the NogoA derived peptide, NEP1 -40, even though both reagents successfully promote neurite outgrowth or axonal regeneration in vitro and in vivo. Recently, two COPYRIGHT (c) 2007 The Thomson Corporation ANSWER 9 OF 25 BIOSIS L14 AB.

homologues,.

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DRG neurites; DRG neurons: nervous system; nervous system CNS: nervous system; Ħ

myelin; IN-1; MAG; NEP1-40; Chemicals & Biochemicals

NogoA; antibodies, leucine; myelin protein; p75; rhoA Nogo; Nogo6;

- ANSWER 10 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation L14
- 66 and MAG in CHO cells (2002, Science 297:1190-3) showed competitive binding between AP-Nogo66 and MAG in CHO cells overexpressing NgR1, whereas Liu et al. (2002, Science 297:1190-3) shot that ARP1-40 (a peptide derived from Nogo66) was unable to block MAG binding to NgR1 but successfully blocked AP-Nogo66. We compared MAG. AB.

IT . . Organisms

CNS: nervous system; membrane; neurons: nervous system;

oligodendrocytes: nervous system Chemicals & Biochemicals

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GPI; GST; MAG [myelin-associated glycoprotein]; MEP1-40; Nogo-A; Nogo66; SPA; glycoprotein; membrane protein; myelin

- ANSWER 11 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation STN 114
- terminal fragments of the Nogo-66 domain. This antagonist binds to the Nogo-66 Receptor. These findings reveal the central role of the Nogo-66 Receptor in limiting axonal regeneration after adult mammalian CNS injury, and NEPL-40 provides a potential therapeutic approach to treating traumatic CNS axonal injury. conclusion about the role of Nogo-66 or its receptor (NgR). We identify a peptide antagonist (Nogo Extracellular Peptide residues 1-40, NEP1-40) of the Nogo-66 Receptor derived from amino ġ

nervous system

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Diseases H

spinal cord injury: injury, nervous system disease, drug therapy Spinal Cord Injuries (MeSH) H

NEP1-40 [NOSO extracellular peptide residues 1-40]: central stimulant-drug, neuroprotectant-drug, pharmacodynamics; Nogo-66 receptor: therapeutic recovery; myelin Chemicals & Biochemicals

- 6 COPYRIGHT (c) 2007 The Thomson Corporation ANSWER 12 OF 25 BIOSIS 14
- approach can be adapted to systemic therapy in a postinjury therapeutic time window. Subcutaneous treatment with the NgR antagonist peptide NTP1-40 (Nogo extracellular peptide, residues 1-40) results in extensive growth of corticospinal axons, sprouting of serotonergic fibers, upregulation of axonal growth. protein IA), and synapse re-formation. Locomotor recovery after thoracic spinal cord injury is enhanced. Furthermore, delaping the initiation of systemic NRB1-40 administration for up to 1 week after cord lessons does not limit the degree of axon sprouting and functional ė,

serotonergic fiber: nervous system Diseases

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spinal cord injury: injury, nervous system disease Spinal Cord Injuries (MeSH)

Chemicals & Biochemicals 片

NZP1-40 peptide; Nogo; Nogo-66 receptor antagonist: delayed systemic administration; SPRRIA [small proline-rich repeat

protein 1A]

ANSWER 13 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation L14

from antino-terminal peptide fragments of Nogo-66. The Nogo-66(1-40) antagonist peptide (NZP1-40) blocks Nogo-66. The Nogo-66(1-40) antagonist peptide (NZP1-40) blocks Nogo-66 or CNS myelin inhibition of axonal outgrowth in vitro, demonstrating that NgR mediates a significant portion of axonal outgrowth inhibition by myelin. Intrathecal administration of NZP1-40 to rats with mid-thoracic spinal cord hemisection results in significant axon growth of the corticospinal tract, and improves functional recovery. Thus, Nogo-66 and NgR have central roles in limiting axonal regeneration after CNS injury, and NZP1-40 provides a potential therapeutic AB.

L14 AB

ANSWER 14 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN

the thalamus in RHRSP was observed at 1, 2 and 4 wk after distal MVAO. In addition, intracerebroventricular infusion of NEP140, a Nogo-66 receptor (NGR) antagonist peptide, was administered starting 24 h after MCAO and continued for 1, 2 and 4.

starting 24 h after MCAO and continued for 1, 2 and 4. the expression of Nogo-A in oligodendrocytes increased persistently and its localization became redistributed around damaged axons and dendrites. Administration of NEP1-40 downregulated the expression of Nogo-A, reduced axonal injury and enhanced axonal regeneration. Our data suggest that Nogo-A is involved in.

(axon; NEP1-40 reduced axonal injury and enhanced axonal regeneration)

Axon H

Axon

H

Injury

II

(disease, injury; NEP1-40 reduced axonal injury and enhanced axonal regeneration)

(regeneration, NEP1-40 reduced axonal injury and enhanced axonal regeneration)

COPYRIGHT 2007 ACS on STN CAPLUS ANSWER 15 OF 25 11 TI AB

Cloning of NEP1.40 gene and expression of its protein

The genes of nogo-66 and NEP1.40 from spinal cord of
rat were cloned and the expression of its protein in vitro was studied.

The nogo-66 and NEP1.40 genes were cloned from the
The nogo-66 and NEP1.40 genes were cloned from the
spinal cord of juvenil rat by RT-PCR techniques, and the objective genes
were bonded to.

Ni-column and detected by using Western-blot test. Results showed that the Nogo-66 and NEP1-40 genes were successfully cloned from rat, which were 215 bp and 137 bp for each one when add the enzymethe results of electrophoresis. The expression plasmids were

in vitro using genetic engineering, which provided a good It was conclusion that the results of electrophoresis. The expression plasmids were induced by IPTG and got the purified GST fusion protein nogo-66 and NEPL-40, which relative mol. weight were 33.2+103 and 30.3+103, resp. The results of Western-blot test confirmed that the antigenicity of the two proteins was precise. It was conclusion that Nogo-66 and NEPL-40 proteins could be expressed in basis for further research.

glycoprotein) and OMgp (oligodendrocyte-myelin glycoprotein) ord injury (SCI) and the immunotherapy for SCI with monoclonal ANSWER 16 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN L14 AB

antibodies of Nogo-A, NEP1-40 and DNA vaccines, etc. immunotherapy for

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inhibitors (MAI). To overcome CSPG- or myelin-induced inhibition, strategies based on extrinsic and intrinsic treatments have been developed. For example, MEPI-40 is a synthetic

peptide that promotes axonal regeneration by blocking Nogo-66/NgR interaction and chondroitinase ABC (ChABC), which degrades CS, thereby. Immodel, overexpressed CSPG and MAI impaired axon regrowth, which mimics regeneration failure in vivo. Both CS cleavage with ChABC and MEPI-40 strongly facilitated the regrowth of entorhinal axons after axotomy, permitting the re-establishment of synaptic contacts with target cells. However, the combined treatment did not improve the regeneration induced by ChABC alone, and the delayed treatment of ChABC, but not MEPI-40, had a less pronounced effect on insight into the development of. . . . regeneration entorhino hippocampal axon degrdn proteoglycan signaling; These results provide regrowth compared with acute treatment.

chondroitinase ABC NEP1 40 entorhino hippocampal axon regeneration

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ANSWER 18 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN
A review. IN-1, the monoclonal antibody against the exon 3-encoded
N-terminal domain of Nogo-A, and the Nogo-66 receptor (NgR) antagonist
MEDI-40 have both shown efficacy in promoting
regeneration in animal spinal cord injury models, the latter even when
administered s.c. 1. . . targeted disruption of Nogo and NgR have, . administered s.c. 1. targeted disruption of Nogo and NgR have, surprisingly, only modest regenerative capacity (if any) compared with treatment with IN-1 or NEP1-40. Disruption of the Nogo gene by various groups yielded results ranging from significant regenerative improvement in young mice to number background, suggest here some possible and testable explanations for the above phenomena. These possiblities include effects of IN-1 and NEP1 -40 on the CNS beyond neutralization of Nogo and Nog functions, and the latter's possible role in the CNS beyond that. ₽ E

therapeutic time window. S.C. treatment with the NgR antagonist peptide MPR-40 (Mogo extracellular peptide, residues 1-40) results in extensive growth of corticospinal axons, sprouting of serotonergic fibers, upregulation of axonal growth. . protein 1A), and synapse re-formation. Locomotor recovery after thoracic spinal cofiniury is enhanced. Furthermore, delapting the initiation of systemic MPR1-40 administration for up to 1 wk after cord lesions does not limit the degree of axon sprouting and functional recovery.. . CAPLUS COPYRIGHT 2007 ACS on STN ANSWER 19 OF 25 L14 AB

ANSWER 20 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN

or NGR. Here, we identify competitive antagonists of NGR derived
from amino-terminal peptide fragments of Nogo-66. The Nogo-66 (1-40)
antagonist peptide (NRF1-40) blocks Nogo-66 or CNS
myelin inhibition of axonal outgrowth in vitro, demonstrating that NGR
mediates a significant portion of axonal outgrowth inhibition by myelin.
Intrathecal administration of NRF1-40 to rats with
mid-thoracic spinal cord hemisection results in significant axon growth of
the corticospinal tract, and improves functional recovery. Thus, Nogo-66
and NGR have central roles in limiting axonal regeneration after CNS
injury, and NRF1-40 provides a potential therapeutic agent. R 51

the thalamus in RHRSP was observed at 1, 2 and 4 weeks after distal McMo. In addition, intracerebroventricular infusion of the thalamus in Mrsp-40, a Nogo-66 receptor (NgR) antagonist peptide, was administered starting 24 h after MCMO and continued for 1, 2 and 4. . . the expression of Nogo-A in ollgoedendrocytes increased persistently and its localization became redistributed around damaged axons and dendrites. Administration of NRPI-40 downregulated the expression ANSWER 21 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN L14

AB

therapeutic time window. Subcutaneous treatment with the NgR antagonist peptide MRB1-40 (Nogo extracellular peptide, residues 1-40) results in extensive growth of corticospinal axons, sprouting of serotonergic fibers, upregulation of axonal growth. . protein 1A) and synapse re-formation. Locomotor recovery after thoracic spinal cord

ANSWER 24 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

monoclonal antibody AS472: DT,.

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IN-1, the monoclonal antibody against the exon 3-encoded N-terminal domain Our with of Nogo-A, and the Nogo-66 receptor (NgR) antegonist NEPI-40 have both shown efficacy in promoting regeneration in animal spinal cord injury models, the latter even when administered subcutanceusly 1. : targeted disruption of Nogo and NgR have, surprisingly, only modest regenerative capacity (if any) compared wit treatment with IN-1 or NEPI-40. Disruption of the Nogo gene by various groups yielded results ranging from significant regenerative improvement in young mice to number . . background, w ANSWER 22 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All.rights reserved on STN of Nogo-A, reduced axonal injury and enhanced axonal regeneration. data suggest that Nogo-A is involved in. intracerebroventricular drug administration microtubule associated protein 2: EC, endogenous compound nepl 40: DV, drug development nepl 40: DT, drug therapy nepl 40: CV, intracerebroventricular drug administration nepl 40: PD, pharmacology Nogo 66 receptor amyloid precursor protein: EC, endogenous compound neuromodulin: Ec, endogenous compound "protein Nogo A: Ec, endogenous compound receptor blocking agent: DV, drug development receptor blocking agent: DT, drug. stroke prone spontaneously hypertensive rat . . localization thalamus ventral nucleus (1) Nep1 40 animal . 114 ដ 2

ANSWER 23 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN SC, subcutaneous drug administration suggest here some possible and testable explanations for the above phenomena. These possibilities include effects of IN-1 and MEP1-40 on the CNS beyond neutralization of Nogo and NgR functions, and the letter's possible role in the CNS beyond that. intravenous drug administration pharmacology intraperitoneal drug development drug comparison drug therapy monoclonal antibody NZP1 40: SC, subcutaneous monoclonal antibody AS472: CM, drug comparison monoclonal antibody AS472: DV, drug development drug development drug therapy pharmacology monoclonal antibody 7B12: DV, drug monoclonal antibody 7B12: DT, drug monoclonal antibody 7B12: PV, intramonoclonal antibody 7B12: IV, intramonoclonal antibody MEP1 40: CM, monoclonal antibody NEP1 40: CM, monoclonal antibody NEP1 40: DV, monoclonal antibody NEP1 40: DT, monoclonal antibody NEP1 40: PD, monoclonal antibody NEP1 40: IP, Medical Descriptors: administration *central . comparison 114 ដ

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injury is enhanced. Furthermore, delaying the initiation of systemic NZB1-40 administration for up to 1 week after cord lesions does not limit the degree of axon sprouting and functional recovery..

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. or NGR. Here, we identify competitive antagonists of NgR derived from aminoterminal peptide fragments of Nogo-66. The Nogo-66(1-40) antagonist peptide (NZP1-40) blocks Nogo-66 or CNS myelin inhibition of axonal ouegrowth in vitro, demonstrating that NgR mediates a significant portion of axonal ouegrowth inhibition by myelin. Intrathecal administration of MZP1-40 to rats with midthoracic spinal cord hemisection results in significant axon growth of the corticospinal tract, and improves functional recovery. Thus, Nogo-66 and NgR have central roles in limiting axonal regeneration after CNS singury, and MZP1-40 provides a potential therapeutic

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ANSWER 1 OF 25 MEDLINE on STN
200723940 IN-PROCESS
PubMed ID: 17382469
NOGO-A is involved in secondary axonal degeneration of thalamus in hypertensive rats with focal cortical infarction.
Nypertensive rats with focal cortical infarction.
Nang Fang; Liang Zhijian; Hou Qinghua; Xing Shihui; Ling Li, He Meixia; Pei Zhong; Zeng Jinsheng
Pei Zhong; Zeng Jinsheng
Show Yar-Sen University, No. 58 Zhongshan Road 2, Guangzhou 510080, China.
Neuroscience letters, (2007 May 7) Vol. 417, No. 3, pp. 255-60.
Electronic Publication: 2007-03-12.

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Journal, Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T) English Ireland 급성

NONMEDIJUB; IN-PROCESS; NONINDEXED; Priority Journals Entered STN: 24 Apr 2007 Last Updated on STN: 13 Jun 2007 E ES

MEDLINE on STN ANSWER 2 OF 25

MEDLINE 2006120738

PubMed ID: 16407455 TANE

Regeneration of lesioned entorhino-hippocampal axons in vitro by combined degradation of inhibitory proteoglycans and blockade of Nogo-66/NgR

Mingurance Ana; Sole Marta: Muneton Vilma; Martinez Albert; Nieto-Sampedro Mingueance Ana; Sole Marta; Dose Antonio Development and Regeneration of the CNS, Department of Cell Biology, IRB-PCB, University of Barcelona, Barcelona, Spain.
The FASEB journal: official publication of the Pederation of American Societies for Experimental Biology, (2006 Mar) Vol. 20, No. 3, pp. 491-3. Electronic Publication: 2006-01-11.
Journal code: 8804484. E-ISSN: 1530-6860.
(IN VITRO) AU CS

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Journal, Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)

Priority Journals

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200604 Entered STN: 2 Mar 2006 Last Updated on STN: 22 Apr 2006 Entered Medline: 21 Apr 2006

MEDLINE on STN IN-PROCESS ANSWER 3 OF 25 L14 AN TI AU CS

PUDGEOTISEZ IN-PROCESS
PubMed ID: 16457436
Cloning of MEP1-40 gene and expression of its protein.
Gong Fuliang; Wang Kunzheng; Yu Pengbo
Department of Orthopseedic Surgery, Second Hospital of Xi'an Jiaotong
University, Xi'an Shaanxi, 710004, PR China.
Zhongguo xiu fu chong jian wai ke za zhi = Zhongguo xiufu chongjian waike
zazhi = Chinase journal of reparative and reconstructive surgery, (2006
Jan) Vol. 20, No. 1, pp. 9-12. S

(ENGLISH ABSTRACT)

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Journal; Article; (JOURNAL ARTICLE)

Chinese NOWMEDLINE, IN-PROCESS, NONINDEXED, Priority Journals Entered STW: 7 Feb 2006 Last Updated on STW: 12 Dec 2006

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ANSWER 4 OF 25 MEDLINE on STN
2005427718
PubMed ID: 16092935
PubMed ID: 16092915
regeneration compared to treatment with neutralizing agents?
Teng Felicia Yu Hsuan; Tang Bor Luen
Department of Biochemistry and Programme in Neurobiology and Aging,
National University of Singapore, Singapore.
Journal of neurochemistry, (2005 Aug) Vol. 94, No. 4, pp. 865-74. Ref: 70 Journal code: 29851908. ISSN: 0022-3042.
England: United Kingdom
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Journal, Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)

General Review, (REVIEW) English

Priority Journals 200509 BERE

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MEDLINE on STN MEDLINE ANSWER 5 OF 25

2003241538 MEDL Pubmed ID: 12764110

Delayed systemic Nogo-66 receptor antagonist promotes recovery from spinal TI DNA

Department angress treptor entagoner promotes recovery trems of shuxin, Strittmatter Stephen M
Li Shuxin, Strittmatter Stephen M
Li Shuxin, Strittmatter Stephen M
Department of Neurology and Section of Neurobiology, Yale University School of Medicine, New Haven, Connecticut 06520, USA.
The Journal of neuroscience: the official journal of the Society for Neuroscience, (2003 May 15) Vol. 23, No. 10, pp. 4219-27.
Journal code: 8102140. E-ISSN: 1529-2401.
Onlied States
Journal; Article; (JOURNAL ARTICLE)
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ANSWER 9 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN ANSWER 7 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN ANSWER 8 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN Univ Barcelona, IRB PCB, Dept Cell Biol, Dev and Regenerat CNS, Barcelona Sci Pk,Josep Samitier 1-5, B-08028 Barcelona, Spain jario@pcb.ub.es Regeneration of lesioned entorhino-hippocampal axons in vitro by combined degradation of inhibitory proteoglycans and blockade of Nogo-66/NgR Why do Nogo/Nogo-66 receptor gene knockouts result in inferior regeneration compared to treatment with neutralizing agents?. Hsuan, Felicia Yu, Tang, Bor Luen [Reprint Author] Natl Univ Singapore, Dept Biochem, 8 Med Dr, Singapore 117597, Singapore Mingorance, Ana, Sole, Marta; Muneton, Vilma; Martinez, Albert; Nieto-Sampedro, Manuel; Soriano, Eduardo; del Rio, Jose Antonio (Reprint Nogo-66 receptor antagonist peptide promotes axonal regeneration. GrandPre Tadzia, Li Shuxin, Strittmatter Stephen M Department of Neurology and Section of Neurobiology, Yale University School of Medicine, New Haven, CT 06520, USA.

Nature, (2002 May 30) Vol. 417, No. 6888, pp. 547-51.

Journal code: 0410462. ISSN: 0028-0836. bchtbl@nus.edu.sg Journal of Neurochemistry, (AUG 2005) Vol. 94, No. 4, pp. 865-874. CODEN: JONRA9. ISSN: 0022-3042. FASEB Journal, (JAN 2006) Vol. 20, No. 1. CODEN: FAJOEC. ISSN: 0892-6638. England: United Kingdom Journal, Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T) (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.) U.S. GOV'T, P.H.S.) General Review; (Literature Review) MEDLINE on STN 200206 Entered STN: 31 May 2002 Enter Updated on STN: 28 Jun 2002 Entered Medline: 27 Jun 2002 Entered STN: 24 May 2003 Last Updated on STN: 26 Jun 2003 Entered Medline: 25 Jun 2003 English Entered STN: 7 Jun 2006 Last Updated on STN: 7 Jun 2006 Entered STN: 26 Oct 2005 Last Updated on STN: 26 Oct 2005 2002297070 MEDLINE Pubmed ID: 12037567 BIOSIS BIOSIS (RESEARCH SUPPORT, English Priority Journals Priority Journals 2005:440461 BIO PREV20051022444 PREV200600300324 6 OF 25 2006:305359 signaling. Article English 200306 TO ALL LI14 DN TII AU CS 8 19 BESE A S 8 658 E E E ដូជ

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Do MoG and Nogode Compete for binding to NgR1?

Jirik, A. P. [Reprint Author]; Li, W. [Reprint Author]; Pepinsky, B. [Reprint Author]; Wang, X. [Reprint Author]; Wang, X. [Reprint Author]; Sah, D. W. Y. [Reprint Author]; Lee, D. H. S. [Reprint Author]; Rabachi, S. A. [Reprint Author]; Lee, D. H. S. NeuroBiol., Cambridge, MA, USA

Revorant Author]; Rabachi, S. A. [Reprint Author]

NeuroBiol., Cambridge, MA, USA

Society for NeuroScience Abstract Viewer and Itinerary Planner, (2003)

Vol. 2003, pp. Abstract No. 678.2. http://sfn.scholarone.com.e-file.

Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Confeanse, (Meeting)

Conference, (Meeting)
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NOGO - 66 RECEPTOR ANTACONIST PEPTIDE PROMOTES AXONAL REGENERATION AND NOGO - 66 RECEPTOR ANTACONIST PEPTING CORD INJURY.

Li, S. (Reprint Author), Grandbre, T. (Reprint Author), Strittmatter, S. M. (Reprint Author)
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Society for Neuroscience Abstract Viewer and Itinerary Planner, (2002)
Vol. 2002, pp. Abstract No. 203.4. http://sfn.scholarone.com. cd-rom.
Meeting Info: a 3nd Annual Meeting of the Society for Neuroscience.
Orlando, Florida, USA. November 02-07, 2002. Society for Neuroscience.
Conference; (Meeting)
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Myelin-associated inhibitive molecules and immune therapy for spinal cord Delayed systemic Nogo-66 receptor antagonist promotes recovery from spinal Nogo-A is involved in secondary axonal degeneration of thalamus in hypertensive rats with focal cortical infarction hypertensive rats with focal cortical infarction Manay, Fangy Liang, Zhijian, Hou, Qinghua, Xing, Shihui, Ling, Li, He, Meixia, Pei, Zhong, Zeng, Jinsheng Department of Neurology and Stroke Center, The First Affillated Hospital, Neuroscience Letters 17, Guangzhou, 510080, Peop. Rep. China CODEN: NELEDS; ISSN: 0104-1940 ខ GrandPre, Tadala, Li, Shuxin, Strittmatter, Scephen M. [Reprint author]
Department of Neurology and Section of Neurobiology, Yale University
School of Medicine, P.O. Box 208018, New Haven, CT, 06520, USA
stephen. strittmatter@yale.edu
Nature (London), (30 May, 2002) Vol. 417, No. 6888, pp. 547-551. print.
CODEN: NATUAS. ISSN: 0028-0836. stephen.strittmatter@yale.edu Journal of Neuroscience, (May 15 2003) Vol. 23, No. 10, pp. 4219-4227. Cloning of WZP1-40 gene and expression of its protein.

Gong, Fuliang, Wang, Kunzheng; Yu, Pengbo, Dang, Xiaoqian; Wang, Chunsheng; Shi, Zhibin; Yang, Pei
The Second Hospital, Xian Jaotong University, Xian, Shanxi Province, 710004, Peop. Rep. China
Zhongquo Xiufu Chongjian Waike Zazhi (2006), 20(1), 9-12
Sichuan Daxue Huaxi Yiyuan Department of Neurology and Section of Neurobiology, Yale University School of Medicine, P.O. Box 208018, New Haven, CT, 06520, USA ANSWER 13 OF 25 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation Nogo-66 receptor antagonist peptide promotes axonal regeneration. THERE ARE 18 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT Stephen M. [Reprint Author] ANSWER 15 OF 25 CAPLUS COPYRIGHT 2007 ACS on STN 2007:263346 CAPLUS CAPLUS COPYRIGHT 2007 ACS on STN Entered STN: 14 Aug 2002 Last Updated on STN: 14 Aug 2002 Entered STN: 19 Jun 2003 Last Updated on STN: 19 Jun 2003 0270-6474 (ISSN print). Li, Shuxin; Strittmatter, 2002:430039 BIOSIS ANSWER 14 OF 25 CA: 2007:448022 CAPLUS Elsevier Ltd. Chinese English English print. PB E1: DT JOI LLA En: L14 AN LIA AN TI AN NOT L14 ပ္သ NA T P S 658 AU SO S E 13 TI AN S BEA SO

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Delayed systemic Nogo-66 receptor antagonist promotes recovery from spinal
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Yin, Guodong, Tang, Xun
Department of Orthopsedics, Kunming General Hospital of Chengdu Military
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JOURNAL: General Review
                                                                                                                                                                                                                                                                                                                                                                                                                                                      Mingorance, Ana; Sole, Marta; Muneton, Vilma; Martinez, Albert; Mingorance, Ana; Sole, Marta; Muneton, Vilma; Martinez, Albert; Misto-Sampedro, Manuel; Soriano, Eduardo; del Rio, Jose Antonio Development and Regeneration of the Central Nervous System (CNS), Department of Cell Biology, Barcelona Science Park, University of Barcelona, Barcelona, 08028, Spain 10.1096/fj.05-5121fje CODEN: FANDEC; ISSN: 0892-6638 Federation of American Societies for Experimental Biology
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                                                         Command, Kunming, 650032, Peep. Rep. China
Zhonghua Chuangshang Zazhi (2005), 21(7), 551-553
CODEN: ZCZAPD, 15SN: 1001-8050
Journal, General Review
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CODEN: JNRSDS; ISSN: 0270-6474
Society for Neuroscience
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2002:403264 CAPLUS
137:362909
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                                                                                                                                                                                                                                                                                                                                                                    144:445304
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Strategies for developing Nogo antagonists.
Prinjha R.K.; McAdam R.A.; Burbidge S.A.; Ellis J.H.
R.K. Prinjha, Neurology and GI-CEDD, New Frontiers Science Park, Third ANSWER 21 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN 2007183702 EMBASE NOGO-A is EMBASE NOGO-A is involved in secondary axonal degeneration of thalamus in hypertensive rats with focal cortical infarction. Hypertensive rats with focal cortical infarction. Hypertensive rats with focal cortical infarction.

A. Zeng, Department of Neurology, Stroke Center, The First Affiliated Hospital, No. 58, Zhongshan Road 2, Guangzhou, 510080, China. TEGERY OF STATE OF THE STATE OF Nogo-66 receptor antagonist peptide promotes axonal regeneration GrandPre, Tadaia; Li, Shuxin; Strittmatter, Stephen M. Department of Neurology and Section of Neurobiology, Yale University School of Nedicine, New Haven, CT, 06520, USA Nature (London, United Kingdom) (2002), 417(6888), 547-551 (CDER: NATUAS; ISSN: 0028-0836 Nature Publishing Group Dournal English ANSWER 23 OF 25 EMBASE COFYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN 2005069346 EMBASE ANSWER 22 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights zengjs@pub.guangzhou.gd.cn Neuroscience Letters, (7 May 2007) Vol. 417, No. 3, pp. 255-260. THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS RECORD ALL CITATIONS AVAILABLE IN THE RE FORMAT Journal, Article
029 Clinical Biochemistry
037 Drug Literature Index
005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery Journal, (Short Survey)
008 Neurology and Neurosurgery
022 Human Genetics English Entered STN: 31 May 2007 Last Updated on STN: 31 May 2007 Entered STN: 22 Sep 2005 Last Updated on STN: 22 Sep 2005 Refs: 18 ISSN: 0304-3940 CODEN: NELEDS S 0304-3940(07)00245-5 Refs: 71 ISSN: 0022-3042 CODEN: JONRA United Kingdom Drug Literature Index English reland English English 14 Ę, 114 L14 L14 AN TI PUI CY DI FS S TI AU CS E E E E AN II CS S BSE AN TI AQ CS တ္ထ CY DT FS 8 S E

2003321746 EMBASE Delayed systemic Nogo-66 receptor antagonist promotes recovery from spinal Dr. S.M. Strittmatter, Department of Neurology, Section of Neurobiology, Atle University School of Medicine, D.O. Box 208018, New Haven, CT 06520, United States. Stephen. Strittmatters/yale.edu Journal of Neuroscience, (15 May 2003) Vol. 23, No. 10, pp. 4219-4227. reserved on STN
1002199201 EMBASE
1002190201 EMB ANSWER 24 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights ANSWER 25 OF 25 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights s alzheimer and (axonal (w)regeneration) 63 ALZHEIMER AND (AXONAL (W) REGENERATION) Neurology and Neurosurgery Human Genetics Clinical Biochemistry Neurology and Neurosurgery Drug Literature Index Neurology and Neurosurgery English Entered STN: 24 Feb 2005 Last Updated on STN: 24 Feb 2005 English Entered STN: 28 Aug 2003 Last Updated on STN: 28 Aug 2003 Entered STN: 20 Jun 2002 Last Updated on STN: 20 Jun 2002 Refs: 50 ISSN: 0270-6474 CODEN: JNRSDS Refs: 14 ISSN: 0028-0836 CODEN: NATUAS Drug Literature Index Drug Literature Index Li S.; Strittmatter S.M. S 1740-6773 (04) 00016-6 United Kingdom Journal; Article Pharmacology Journal; Article Journal; Article Pharmacy 1740-6773 reserved on STN United Kingdom United States cord injury. English English English PUI CY DT FS 114 174 BSE A I S S S CY DT FS BSE SATAS S 323 ម្មន្ន

Yale

Avenue, Harlow, Essex CM19 5AW, United Kingdom. Rabinder Prinjha-1@gsk.com Drug Discovery Today: Therapeutic Strategies, (2004) Vol. 1, No. 1, pp.

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=> d 117 58-63

A movel neurotrophic agent, T-817WA [1-{3-[2-(1-benzothiophen-5-y1)ethoxy]propyl}-3-azetidinol maleate], attenuates amyloid- β -induced neurotoxicity and promotes neurite outgrowth in rat cultured central ANSWER 58 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN 2005:193359 TOXCENTER Copyright 2007 ACS CA14314241806U L17 AN CP DN ΑŪ

nervous system neurons
Hirata, Kazunari; Yamaguchi, Hidetoshi; Takamura, Yusaku; Takagi, Akiko;
Fukushima, Tetsuo; Iwakami, Noboru; Saitoh, Akihito; Nakagawa, Masaya;
Yamada, Tatsuo
Research Laboratories, Toyama Chemical Co., Ltd., Toyama, Japan.
Nournal of Rharmacology and Experimental Therapeutics, (2005) Vol. 314,
CODEN: Upp. 252-259
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80 83

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Entered STN: 19 Jul 2005 Last Updated on STN: 29 Aug 2006 CAPLUS 2005:603092 English

ANSWER 59 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN

2005:154827 TOXCENTER Copyright (c) 2007 The Thomson Corporation PREV200500203216 L17 AN CP DN TI

Neuroprotective role of testosterone in the nervous system Bialek, Magdalena; Zaremba, Pawel; Borowicz, Kinga K.; Czuczwar, Stanislaw J. [Reprint Author]
Dept Pathophysiol, Skubiszewski Med Univ, Jaczewskiego 8, PL-20090, Lublin, Poland czuczwarsjewyahoo.com
Polish Journal of Pharmacology, (September 2004) Vol. 56, No. 5, pp. S

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509-518. print. ISSN: 1230-6002.

General Review; (Literature Review) H

BIOSIS 2005:211699 English

Entered STN: 7 Jun 2005 Last Updated on STN: 7 Jun 2005 SSAG

ANSWER 60 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN 2005:92303 TOXCENTER COpyright 2007 ACS CA14221386101Z A TING CE

Neuroprotective role of testosterone in the nervous system Bialek, Magdalena; Zaremba, Pawel; Borowicz, Kinga K.; Czuczwar, Stanislaw

Department of Pathophysiology, Skubiszewski Medical University, Lublin, PL (2004) Vol. 56, No. 5, pp. 509-518. 20-090, Pol... Polish Journal of Pharmacology, CODEN: PJPAE3. ISSN: 1230-6002. 8 S

Journal POLAND

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CAPLUS CAPLUS 2005:243139

English Entered STN: 22 Mar 2005 Last Updated on STN: 29 Nov 2005

ANSWER 61 OF 63 TOXCENTER COPYRIGHT 2007 ACS ON STN 2003:55704 TOXCENTER COPYRIGHT 2007 ACS L17 AN CP DN TI

CA13812162764X

Recent advance in adenoviral gene transfer technology for neuronal survival and axonal regeneration
Namikawa, Kazuhiko; Kiyama, Hiroshi
Dep, Anatomy, Grad. Sch. Med., Osaka City Univ., Japan.
Saishin Igaku, (2002) Vol. 57, No. 7, pp. 1591-1600.
CODEN: SAIGAK. ISSN: 0370-8241.

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JAPAN

Journal CAPLUS CAPLUS 2002:604210

CY DT FS OS ED ED

Japanese

Entered STN: 11 Mar 2003 Last Updated on STN: 21 Feb 2006

ANSWER 62 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN
1997:28867 TOXCENTER
PubMed ID: 901730
Postnatal retinal ganglion cells in vitro: protection against reactive coxygen species (ROS) induced axonal degeneration by cocultured astrocytes Lucius R; Sievers J
Anatomisches Institut, Universitat Kiel, Germany
Brain research, (1996 Dec. 16) Vol. 743, No. 1-2, pp. 56-62.
Journal code: 0045503. ISSN: 0006-8993. TI AN II AU CS SO

Netherlands

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Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)

MEDLINE 97169682 MEDLINE

E P E S

English

Entered SIN: 16 Nov 2001 Last Updated on SIN: 16 Nov 2001

ANSWER 63 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN 1996:218505 TOXCENTER

1996:218505

Copyright 2007 ACS

CAISO70857822

Postnatal retinal ganglion cells in vitro: protection against reactive oxygen species (ROS)-induced axonal degeneration by cocultured astrocytes Lucius, Ralph; Sievers, Jobst Anatomisches Institut der Universitaet Kiel, Olshausenstr. 40, Kiel, TI AN TI

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(1996) Vol. 743, No. 1,2, pp. 56-62. D-24118, Germany.

Brain Research, (1996) Vol. 743, CODEN: BRREAP. ISSN: 0006-8993. GERMANY, FEDERAL REPUBLIC OF

CAPLUS 1996:736899

ELS PR

English Entered STN: 16 Nov 2001 Last Updated on STN: 18 Jun 2002

=> d 117 1-63 kwic

ANSWER 1 OF 63 MEDLINE on STN
The dogma that the adult central nervous system (CNS) is nonpermissive
axonal regeneration is beginning to fall in the face of
increased understanding of the molecular and cellular biology of axon
ouggrowth. It. . . . These vectors may be useful in regenerative
strategies for spinal cord injury, brain injury, and neurodegenerative L17

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diseases including Parkinson's disease, Alzheimer's disease, and Huntington's disease.

MEDLINE on STN ANSWER 2 OF 63 L17

also be involved in the pathological processes in diseases such as epilepsy, stroke and Alzheimer's disease. Several possible methods of manipulating CSPGs in the CNS have recently been identified. The development of methods to remove. injuries and diseases. After CNS injury, CSPGs are the minhibitory component of the glial scar. Removal of CSPGs improves axonal regeneration and functional recovery. CSPGs may

7 E

ANSWER 3 OF 63 MEDLINE on STW (RTW) proteins are localized to the endoplasmic reticulum (ER), and are related to intracellular membrane trafficking, apoptosis, inhibiting axonal regeneration, and Alzheimer, as disease. The RTW proteins are produced without an N-terminal signal peptide. Their C-terminal domain contains two long hydrophobic segments. peptide. We. . .

MEDLINE on STN ANSWER 4 OF 63 117 AB

Acons fail to regenerate in the adult central nervous system (CNS) following injury. Developing strategies to promote axonal regeneration is therapeutically attractive for various CNS pathologies such as traumatic brain injury, stroke and Alzheimer 's disease. Because the RhoA pathway is involved in neurite outgrowth, Rho-associated kinases (ROCKS), downstream effectors of GTP-bound Rho, are

4 E

ANSWER 5 OF 63 MEDLINE on STN Progressive neuronal loss in Alzheimer's disease (AD) is. considered to be a consequence of the neurotoxic properties of amyloid-beta peptides (A beta). T-817MA (I-{3·[2·(1-benzothiophen-5-yl)} ethoxy). . . agent for the treatment of AD based on its neuroprotective potency against A beta-induced neurotoxicity and its effect of enhancing axonal regeneration in the sciattc nerve axotomy model.

The neuroprotective effect of T-817MA against A beta(1-42) or oxidative stress-induced neurotoxicity was assessed.

MEDLINE on STN ANSWER 6 OF 63

testosterone actions is neuroprotection. There are some vidences supporting the hypothesis that testosterone may act protectively in neurodegenerative disorders, e.g. Alshaimar's disease (AD), mild cognitive impairment (MCI) or depression. Androgens alter also the morphology, survival and axonal regeneration of motor neurons. These hormones accelerate the regeneration of hamster facial nerve and anterior tibialis sciatic nerve in rabbits following. L17 AB

Modulation of axonal regeneration in neurodegenerative MEDLINE on STN ANSWER 7 OF 63 117

disease: focus on Nogo.

Recent work has demonstrated that axonal regeneration
in the central nervous system is limited by myelin-derived Nogo binding to
an axonal Nogo Receptor. The Nogo system appears.

The Nogo system contributes to pathologic and compensatory plasticity in 2

Alzbeimer's Disease is considered. Alzbeimer Disease: MB, metabolism

Alsheimer Disease: PA, pathology

b

Animals

Axons: ME, metabolism
*Axons: PA, pathology
*Growth Inhibitors: ME, metabolis
*Myelin Proteins: ME, metabolism

estrogen in neuroprotection. Accumulated clinical evidence augusts: that estrogen exposure decreases the risk and delays the onset and progression of Altabaimar's disease and schizophrenia, and may also enhance recovery from traumatic neurological injury such as stroke. Recent basic science studies show. the classical nuclear estrogen receptor, through which estrogen alters expression of estrogen responsive genes that play a role in apoptosis, axomal regeneral trophic support. Yet another possibility is that estrogen receptors in the membrane or cycoplasm alter MEDLINE on STN phosphorylation cascades through. ANSWER 8 OF 63 L17 AB

MEDLINE on STN ANSWER 9 OF 63 L17

Reactive oxygen species (ROS) are supposed to be involved in neurodegenerative processes like Parkinson's or Alzhedmer's disease. Beside this there are an increasing number of studies indicating an involvement of ROS in traumatic brain injury. We astrocytes are able to protect retinal ganglion cells against ROS-induced oxidative stress, (ii) astrocytes release soluble neurotrophic factors supporting RGC axonal regeneration, and (iii) free radical production after tissue injury may partly contribute to the failure of axonal regeneration in the adult mammalian central

nervous system.

ANSWER 10 OF 63 MEDLINE on STN
Aberrant GAP-43 gene expression in Alzheimer's disease.
GAP-43 is a growth-associated phosphoprotein expressed at high levels in neurons during development, axonal regeneration, and neuritic sprouting. GAP-43 gene expression in mature neurons is probably functionally important for the structural remodeling of synapses as arguired for learning and establishing new memory. The widespread aberrant neuritic growth accompanied by impaired synaptic plasticity in Alzheimer's disease (AD) suggests that abnormal GAP-43 gene expression may contribute to the cascade of neurodegeneration. In the present study, end-stage. A117

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Aged, 80 and over

Aging: ME, metabolism *Alzheimer Disease: GE, genetics Alzheimer Disease: PA, pathology

Blotting, Northern Brain: ME, metabolism Brain: PA, pathology GAP-43 Protein

Gene Expression

Humans

Immunohistochemistry In Situ. MEDLINE on STN ANSWER 11 OF 63

acquired immunodeficiency syndrome, based on regrowing or saving injured neurons. The clinical neurologist will become important in practical applications and research into prolonging neuronal survival and fostering axonal regeneration. Over the coming years, with further research, it is anticipated that patients will be treated with . . . molecular techniques underlying nerve growth are discussed. Possible therapeutic approaches are presented for many neurologic disorders, ranging from stroke to Alzheimer's disease to these or similar modulatory. E17

é ANSWER 12 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation 117

- axonal regeneration, and The RTN proteins are produced without an ide. Their C-terminal domain contains two long inhibiting axonal N-terminal signal peptide. hydrophobic segments. Alzheimer's disease. ¥B
- 6 ANSWER 13 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation 117
- following injury. Developing strategies to promote axonal regeneration is therapeutically attractive for various CNS pathologies such as traumatic brain injury, stroke and Alzheimer's disease. Because the RhoA pathway is involved in neurite outgrowth, Rho-associated kinases (ROCKs), downstream effectors of GTP-bound Rho, are Axons fail to regenerate in the adult central nervous system (CNS) potentially. B
- brain injury: nervous system disease, injury
 - Brain Injuries (MeSH)

H

Diseases Ħ

H

nervous system disease (MeSH) stroke: vascular disease, Cerebrovascular Disorders Diseases

Alzheimer's disease: nervous system disease; behavioral and

- mental disorders Chemicals & Biochemicals H
- RhoA; ROCK; Y-27632: enzyme inhibitor-drug; cofilin: dephosphorylation;
- ទ ANSWER 14 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation 117
- considered to be a consequence of the neurotoxic properties of amyloid-beta peptides (A beta). T-817MA (1-{3-{2-(1-benzothiophen-5-yl).}} agent for the treatment of AD based on its neuroprotective potency against A beta-induced neurotoxicity and its effect of enhancing axonal regeneration in the sciatic nerve axotomy model.

 The neuroprotective effect of T-817MA against A beta(1-42) or oxidative Progressive neuronal loss in Alzheimer's disease (AD) AB
- Organisms II of

stress-induced neurotoxicity was assessed.

- nervous system; glial neuron: nervous system; central nervous system: nervou cell: nervous system; cortical neuron: nervous system Diseases H

 - hydrogen peroxide; growth-associated protein 43; GSH; amyloid-beta: Alzheimer's disease: nervous system disease, behavioral and mental disorders Chemicals & Biochemicals H
- 6 ANSWER 15 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation STN 1,17
- Androgens alter also the neurons. These hormones accelerate the regeneration of hamster facial nerve and anterior tibialis sciatic nerve in rabbits following. supporting the hypothesis that testosterone may act protectively in neurodegenerative disorders, e.g. Alzabamer's disease (AD), mild cognitive impairment (WCI) or depression. Androgens alter also morphology, survival and axonal regeneration of motor is neuroprotection. . testosterone actions Ā
- neuron motor neuror spinal cord nervous system; laryngeal motor nucleus: nervous system; nervous system; pelvic autonomic neuron: nervous system, Diseases

H

H

Altheimer's disease: behavioral and mental disorders, nervous system disease, drug therapy

Alzheimer Disease (MeSH)

H

- behavioral and mental disorders, drug therapy (MeSH) depression: Depression
- mild cognitive impairment: behavioral and mental.
- 6 ANSWER 16 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation 117
- Modulation of axonal regeneration in neurodegenerative TI
 - Recent work has demonstrated that axonal regeneration disease: Focus on Nogo. Æ
- in the central nervous system is limited by myelin-derived Nogo binding tan axonal Nogo Receptor. The Nogo system appears.

 a physiologic role in regulating structural plasticity. The possibility that the Nogo system contributes to pathologic and compensatory plasticity in Alzheimer's Disease is considered.
 - Structures, & Systems of Organisms
- axon: nervous system, plasticity, regeneration; central nervous system: nervous system; neurite: nervous system
 - Diseases

H

- Alzheimer's disease: behavioral and mental disorders, nervous system disease
 Alzheimer Disease (MeSH)
 - Chemicals & Biochemicals

Ħ

Nogo: binding activity; Nogo receptor; myelin

- ANSWER 17 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation 117
- individuals with moderate or severe white matter changes (WMC) and in those with mild or no WMC. Twenty-two patients with Alzabaimer is disease (AD), nine patients with subcortical vascular dementia (SVD), and 20 normal controls were included in the study. The occurrence. ā.
- Medicine, 'Medical Sciences)

H

LI

- Parts, Structures, & Systems of Organisms cerebrospinal fluid: nervous system; white matter: nervous system
 - Alzheimer's disease: behavioral and mental disorders, nervous Diseases
 - system disease
 Alzbeimer Disease (MeSH)
- subcortical vascular dementia: behavioral and mental disorders Diseases H
- beta-amyloid 42: neurofilament protein, tau:. Miscellaneous Descriptors

Biochemicals

Chemicals &

H

- apolipoprotein E E4 allele inheritance, axonal regeneration; white matter changes
- ĕ ANSWER 18 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation
 - Major Concepts Ħ
- Nervous System (Neural Coordination); Pharmacology Parts, Structures, & Systems of Organisms brain: nervous system Diseases II H

H

- Altheimer's type senile dementia: behavioral and mental disorders, nervous system disease Chemicals & Biochemicals TEL:3156 [[(165)-12-deoxy-16-hydroxy-16-methyl-9(0)-methano-delta-6(9alpha)-prostaglandin]: anti-amnesic effect, prostacyclin stable.
- Miscellaneous Descriptors

H

axonal regeneration; learning; memory

(c) 2007 The Thomson Corporation COPYRIGHT ANSWER 19 OF 63 BIOSIS 117

AB

- Reactive oxygen species (ROS) are supposed to be involved in neurodegenerative processes like Parkinson's or Altheimer's disease. Beside this there are an increasing number of studies indicating an involvement of ROS in traumatic brain injury. We. . . astrocytes an involvement of ROS in traumatic brain injury. We. . . astrocytes are able to protect retinal ganglion cells against ROS-induced oxidative stress, (ii) astrocytes release soluble neurotrophic factors supporting RGC axonal regeneration, and (iii) free radical production after tissue injury may partly contribute to the failure of axonal regeneration in the adult mammalian central nervous system.
- ទ ANSWER 20 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation 17
- Aberrant GAP-43 gene expression in Alzheimer's disease. H &
- GAP-43 is a growth-associated phosphoprotein expressed at high levels in neurons during development, axonal regeneration, and neuritic sprouting. GAP-43 gene expression in mature neurons is probably functionally important for the structural remodeling of synapses as required for learning and establishing new memory. The widespread aberrant meuritic growth accompanied by impairer synapsic plasticity in Althaimar's disease (AD) suggests that abnormal GAP-43 gene expression may contribute to the cascade of neurodegeneration. In the present study, end-stage.
- ë ANSWER 21 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation
- Disrupted beta-1-adrenoceptor-G protein coupling in the temporal cortex of H
- a sugmitted reduction in the affinity of. . . effect was attributed to the conversion of high agonist-affinity sites to a lower-affinity state and was not found for the Alzhoimer's disease cases. These data indicate that a disruption of beta-1-adrenoceptor-G protein coupling occurs in the temporal cortex of Alzhoimer's disease patients. patients with Althoimer's disease.

 The efficacy of beta-l-adrenoceptor-G protein coupling was studied in postmortem temporal cortex synaptic membranes from a series of control in postmortem temporal cortex synaptic membranes from a series of control and Althoimer's disease subjects. For the control cases, the non-hydrolysable GTP analogue 5'-guanylylimidodiphosphate (Gpp(NH)p) gave a significant reduction in the affinity of. AB.
 - AIONAL REGENERATION IMPAIRMENT; HYPEREMIA; HYPERGLYCEMIA; SENSORY CONDUCTION VELOCITY; VASA NERVORUM NEUROGENIC Miscellaneous Descriptors

H

- ü ANSWER 22 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation 117
- . The carboxy-terminus of the beta amyloid protein is critical for the seeding of amyloid formation: Implications for the pathogenesis of Alzheimer's disease. TI.
- Deta-1-40, beta-1-42, and beta-1-43), have been identified as the major components of the cerebral amyloid deposits which are characteristic of Alshofmer's disease. Kinetic studies of aggregation by three naturally occurring beta protein variants (beta-1-3), beta-1-40, beta-1-42) and four model peptides (beta-26-39, ġ
 - Miscellaneous Descriptors
 AXONAL REGENERATION; CYCLOSPORIN A;

H

NERVE ANASTOMOSIS; NERVE REPAIR; NERVE TRANSPLANTATION; PERINEURIUM IMMUNOSUPPRESSANT-DRUG;

- 6 ANSWER 23 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation 111
- . axotomized medial septal and diagonal band of Broca neurons

Æ.

selectively and rapidly express JLI. The role of Jun expression in axonal regeneration or neuronal death is discussed.
Miscellaneous Descriptors
RAI AXONAL REGENERATION NEURONAL DEATH PROTEIN

H

ALZHEIMER'S DISEASE TRANSCRIPTION FACTORS

- ANSWER 24 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN injuries and diseases. After CNS injury, CSPGs are the major inhibitory component of the glial scar. Removal of CSPGs improves axonal regeneration and functional recovery. CSPGs may also be involved in the pathol. processes in diseases such as epilepsy, extroke and Alzhaimer's disease. Several possible methods of manipulating CSPGs in the CNS have recently been identified. The development of methods to remove. L17
- 117 AB
- ANSWER 25 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN certain polypeptides and polypeptide fragments of Nogo receptor-1 (NgR1) and Nogo receptor-2 (NgR2) for promoting neurite outgrowth, neuronal survival, and axomal regeneration in CNS neurons. Previous studies have shown that the entire leucine rich repeat (LRR) region of NgRI, including the C-terminal. Typically, the polypeptides and polypeptide fragments of the invention act to block MNGR-mediated inhibition of neuronal survival, neurite outgrowth or axonal regeneration of CNS (central nervous system) neurons by inhibiting signal transduction by the NgR complex. Nogo receptor NgRI NgR2 disulfide structure mutant neurite outgrowth; NgR Nogo receptor signaling inhibition CNS neuron axonal
 - - Alzheimer's disease regeneration

H

ST

- Central nervous system agents Disulfide group
 - Glaucoma (disease) Gene therapy
 - Hearing loss

 - Mammalia
- Molecular cloning Multiple sclerosis Parkinson's disease
- (Nogo receptor (NgR) disulfide structure, NgR signaling inhibiting NgR fragments, mutants, fusion products and genetic constructs, and uses in mediating axonal growth)
 - Central nervous system

H

- (neurons, promoting axonal regeneration in; Nogo receptor (igR) disulfide structure, NgR signaling inhibiting NgR fragments, mutants, fusion products and genetic constructs, and uses in mediating axonal growth)
- COPYRIGHT 2007 ACS on STN ANSWER 26 OF 63 CAPLUS L17 AB
- Reticulon (RTN) proteins are localized to the endoplasmic reticulum (ER), and are related to intracellular membrane trafficking, apoptosis, nihibiting axonal regeneration, and Alzaelmer.

 's disease. The RTN proteins are produced without an N-terminal signal peptide. Their C-terminal domain contains two long hydrophobic segments.
- COPYRIGHT 2007 ACS on STN ANSWER 27 OF 63 CAPLUS E17
- Axons fail to regenerate in the adult central nervous system (CNS) following injury. Developing strategies to promote axoual regeneration is therapeutically attractive for various CNS pathologies such as traumatic brain injury, stroke and Alzheimer s's disease. Because the RhoA pathway is involved in neurite outgrowth, Rho-associated kinases (ROCKS), downstream effectors of GTP-bound Rho, are

potentially.

reducing, preventing, etc.) axonal growth inhibition mediated by such NgR1 Alzheimer's disease
Multine and for screening. ANSWER 28 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN may be used as antagonists to NgR1 ligands and, as such, may be useful in treating subjects in need of axonal regeneration (e.g., for antagonizing (e.g., reversing, decreasing, L17 AB

H

Multiple sclerosis Parkinson's disease

(treating; Nogo receptor 1 (NgR1) functional motifs and peptide mimetics and use as antagonists to NgR1 ligands for antagonizing axonal growth inhibition)

11. TI

ANSWER 29 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN
Protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and
uses in promotion of axonal regeneration
Described are the protein sequences of human, mouse and chicken netrin-G1
ligand NGL-1 and their uses in promoting axonal AB

regeneration. The invention concerns a method of promoting the growth or regeneration of neurons, and treating disease or conditions associated with.

H

Protein motifs (1g-like domain, of NGL-1; protein sequences of human, mouse an chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal

regeneration) H

Protein motifs (PDZ domain, -binding motif, of NGL-1; protein sequences of human, mouse and chicken netrin-Gl ligand NGL-1 and uses in promotion of axonal regeneration)

Ħ

Antibodies and Immunoglobulins
RL: BSU (Biological study, unclassified); BIOL (Biological study) (against NGL-1; protein sequences of human, mouse and chicken netrin-Gl ligand NGL-1 and uses in promotion of axomal

RL: BSU (Biological study, unclassified); BIOL (Biological study) (chimeric, against NGL-1; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal Antibodies and Immunoglobulins H

Antibodies and Immunoglobulins

RL: BSU (Biological study, unclassified); BIOL (Biological study) (fragments, against NGL-1; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal H

regeneration)

H

mouse and Antibodies and Immunoglobulins RL: BSU (Biological study) RL: BSU (Biological study, unclassified); BIOL (Biological study, unclassified); Globulinalized, against NGL-1; protein sequences of human, mouse an chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal

H

Proceins LL. BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (leucine-rich repeat, NGL-1 (netrin-G1 ligand 1); protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion

Deat motifs (protein) (leucine-rich repeat, of NGL-1; protein sequences of human, mouse and chicken netrin-Gl ligand NGL-1 and uses in promotion of axonal Repeat motifs

Proteins H

Ħ

BSU (Biological study, unclassified); BIOL (Biological study) (netrin, G1; protein sequences of human, mouse and chicken netrin-G1 RL: BSU

ligand NGL-1 and uses in promotion of axonal

regeneration

H

· Molecular association (netrin-G1 binding to NGL-1; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

H

Signal peptides
RL: BGV (Biological study, unclassified); BIOL (Biological study)
(of NGL-1: protein sequences of human, mouse and chicken netrin-Gl
ligand NGL-1 and uses in promotion of axonal

regeneration) Axon

H

(outgrowth; protein sequences of human, mouse and chicken netrin-G1 ligand ${\rm NGL-1}$ and uses in promotion of axonal regeneration)

H

Analgesics Anti-*Alzheimer*'s agents

Antiarteriosclerotics

Antiparkinsonian agents (protein sequences of human, mouse and chicken netrin-Gl ligand NGL-1

uses in nerve cell dysfunction)

Gallus domesticus Human

II

Mus musculus

Protein sequences

Axon

H

(protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration)

human, mouse and chicken netrin-Gl (regeneration, protein sequences of human, m ligand NGL-1 and uses in promotion of axonal

regeneration)

H

Altheimer's disease Multiple sclerosis Parkinson's disease

(treatment of; protein sequences of human, mouse and chicken netrin-Gl ligand NGL-1 and uses in nerve cell dysfunction)

875208-52-9 RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(PDZ domain-binding motif of NGL-1, -protein sequences of hu and chicken netrin-G1 ligand NGL-1 and uses in promotion of

human, mouse

H

875717-48-9D, subfragment is claimed 875717-49-0 875717-50-3 RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses) axonal regeneration)

Ħ

(amino acid sequence; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal regeneration

875717-67-2 875717-66-1

H

(unclaimed protein sequence; protein sequences of human, mouse chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal RL: PRP (Properties) regeneration)

and

875612-87-6 H

oŧ

(unclaimed sequence; protein sequences of human, mouse and chicken netrin-G1 ligand NGL-1 and uses in promotion of axonal (Properties)

11.7 A.B.

ANSWER 10 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN
for central nervous system repair, focusing on the therapeutic
use of growth factors to reduce cell loss and to enhance axonal
regeneration in the context of both neurodegenerative and traumatic disorders

Ë	review	NGF	axon	nerve	regeneration	CNS	Alzheimer	disease	
H	Nervous	Sys	ystem,	diseas	e)				

Ħ

Nervous system, disease (amount of the control of control of the control of con

Alzheimer's disease

H

enhance (growth factor can be used to reduce cell loss and to axonal regemeration in animal model with Alzheimer's disease)

Ħ

(growth factor can be used to reduce cell loss and to enhance axonal regeneration in animal model with Parkinson's Parkinson's disease

Multiple sclerosis

片

enhance t t (growth factor can be used to reduce cell loss and axonal regeneration in animal model with multiple sclerosis)

Brain H

(growth factor can be used to reduce cell loss and to enhance axonal regeneration in animal model with neurodegenerative and traumatic disorders) Nerve regeneration

Spinal cord, disease Ħ

(injury; growth factor can be used to reduce cell loss and to enhance axonal regeneration in animal model with spinal cord

injury) Injury 片

(spinal cord; growth factor can be used to reduce cell loss and to enhance axonal regeneration in animal model with

H

spinal cord injury)
9061-61-4, Nerve growth factor
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(Biological study); USES (Uses)
(growth factor can be used to reduce cell loss and to enhance axonal regeneration in animal model with neurodegenerative and traumatic disorders)

OF 63 CAPLUS COPYRIGHT 2007 ACS on STN ANSWER 31 E 27

Progressive neuronal loss in Alzheimer's disease (AD) is considered to be a consequence of the neurotoxic properties of amyloid-\$P\$ peptides (AB). T-817MA was screened as. therapeutic agent for the treatment of AD based on its neuroprotective potency against A\$P\$-induced neurotoxicity and its effect of enhancing axonal regeneration in the sciento nerve axoromy model. The neuroprotective effect of F-817MA against A\$P\$(1-42) or oxidative stress-induced neurotoxicity was assessed using.

H

Alzheimer's disease
Anti-Alzheimer's agents
Oxidative stress, bological
(neuroprotective effects of T-817MA against \$\beta\$-amyloid- and
oxidative stress-induced neurotoxicity in rat cultured central nervous system neurons)

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neurogenesis, neuronal growth and regeneration, neuronal survival, and synaptic plasticity. Like neurotrophic factors, lithium and valpoate promote neurite outgrowth and axonal regeneration in cultured neuronal cells and in injury models utilizing retinal cells, sciatic nerve, and spinal cord. These mood stabilizers also. 8 E

insults and reduce neuronal loss and associated functional deficits in animal models of Alzheimer's disease, HIV-associated encephalitis and dementia, Huntington's disease, ischemia, and Parkinson's disease. Cross-sectional and longitudinal brain imaging studies show that lithium.

Disease models

Ħ

(lithium, valproate showed neurotrophic action by reducing neuronal loss, associated functional deficit via ERK, P13K pathway in animal model or Alzheimer's, Parkinson's, Huntington's disease, ischemia, Alzheimer's disease

II

Ħ

(mood stabilizer lithium and valproate showed neurotrophic action by reducing neuronal loss and associated functional deficit via ERK, P13K pathway activation in animal model of Alzhelmer's disease)

(mood stabilizer lithium, valproate promoted neurogenesis, azonal regeneration, reduced neuronal loss in animal model and increased brain N-acetyl aspartate, cerebral gray matter via ERK, Plik pathway activation in mood disorder patient)

(mood stabilizer lithium, valproate promoted neurogenesis, neurite outgrowth and axonal regeneration via ERK, Pl3K pathway activation in cultured neuronal cell)

ä

H

Neuron

H

axonal regeneration, reduced neuronal loss in animal model and increased brain N-acetyl aspartate, cerebral gray matter via ERK, P13K pathway activation in mood disorder patient)
115926-52-8, Phosphoinositide 3-kinase 142243-02-5, Extracellular signal regulated kinase Nervous system agents (mood stabilizer; lithium, valproate promoted neurogenesis,

RI. BSU (Biological study, unclassified); BIOL (Biological study)

(mood stabilizer lithium, valproate promoted neurogenesis,

axonal regeneration, reduced neuronal loss in animal

model and increased brain N-acetyl aspartate, cerebral gray matter via

ERK, PI3K pathway activation in mood disorder patient)

99-66-1 743-93-22. Lithium, salts

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL

(Biological study); USES (Uses)

(mood stabilizer lithium, valproate promoted neurogenesis,

axonal regeneration, reduced neuronal loss in animal

model and increased brain N-acetyl aspartate, cerebral gray matter via

ERK, P13K pathway activation in mood disorder patient)

H

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testosterone actions is neuroprotection. There is some evid supporting the hypothesis that testosterone may act protectively in neurodegenerative discorders, e.g. Althelmer's discase (AD), mild cognitive impairment (MCI), or depression. Androgens alter also the morphol, survival and axonal regeneration of motor neurons. These hormones accelerate the regeneration of hamster facia nerve and anterior tibialis sciatic nerve in rabbits following. E17

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Modulation of axonal regeneration in neurodegenerative L17 TI

disease. Focus on Nogo A review. Recent work has demonstrated that axonal A review. 2

regeneration in the central nervous system is limited by myelin-derived Nogo binding to an axonal Nogo Receptor. The Nogo system appears.

a physiol. role in regulating structural plasticity. The possibility that the Nogo system contributes to pathol. and compensatory plasticity in Alzheimer's Disease is considered.

review Nogo receptor axon regeneration neurodegeneration Alzhelmer

ST

(Biological study) RL: BSUZ (Biological study, unclassified); BIOL (Biologic (Nogo; Nogo receptor in modulation of axonal regeneration in neurodegenerative disease)
Nervous system, disease
regeneration; Nogo receptor in modulation of axonal regeneration in neurodegenerative disease) Nerve regeneration
Nerve regeneration
Synablic plasticity
(Nogo receptor in modulation of axonal regeneration in neurodegenerative disease) Receptors Proteins H H

for neuronal Recent advance in adenoviral gene transfer technology survival and axonal regeneration ANSWER 35 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN 117 TI

A review. Neuron-targeted gene transfer by adenovirus for the gene therapy of neuronal survival and axonal regeneration in the treatment of Parkinson's disease, Alzheimer's disease, malignant glioma etc. is reviewed. AB

Nerve regeneration Nerve regeneration H

(axonal; recent advance in adenoviral gene transfer technol. for neuronal survival and axonal regeneration) Antitumor agents

(glioma; recent advance in adenoviral gene transfer technol. for neuronal survival and axonal regeneration) Ħ

Anti-Alzheimer's agents Antiparkinsonian agents Adenoviral vectors
Alzheimer's disease Neuroglia, neoplasm II

Parkinson's disease

Transformation, genetic (recent advance in adenoviral gene transfer technol. for neuronal survival and axonal regeneration)

L17 TI Æ

ANSWER 16 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN
Apolipoprotein E and lipid mobilization in neuronal membrane remodeling
and its relevance to Alzhaimer's disease
A review on the link between apolipoprotein E (apoE) to either one of the
two hallmarks of Alzhaimer's disease (AD), namely amyloid plaque
formation and neurofibrillary trangles. It includes a description of
apolipoprotein E (apoE) and its gene, . . the role of apoE as a
modulator of lipid homeostasis and synaptic plasticity. The well
regeneration and remyelination 'involving apoE and LDL receptors is
presented first. The entorhinal cortex lesioning (ECL) model, which

mimics certain neuropathol.. review apolipoprotein E lipid mobilization Alzheimer disease

neuron regeneration H ST

BSU (Biological study, unclassified); BIOL (Biological study) (APOE; apolipoprotein E and lipid mobilization in neuronal membrane remodeling and its relevance to **Althoimer's** disease) animal Gene, H

BSU (Biological study, unclassified); BIOL (Biological study) (E; apolipoprotein E and lipid mobilization in neuronal membrane remodeling and its relevance to Alzheimer's disease) Lipoprotein receptors RL: BSU (Biological st Apolipoproteins RL: BSU (Biologi H

BSU (Biological study, unclassified); BIOL (Biological study) (LDL; apolipoprotein E and lipid mobilization in neuronal membrane

remodeling and its relevance to ${\tt Alzheimer's}$ disease) ${\tt Alzheimer's}$ disease

Nerve regeneration

H

Nerve regeneration Neurofibrillary tangle Synaptic plasticity

(apolipoprotein E and lipid mobilization in neuronal remodeling and its relevance to Alzheimer's disease)

Brain

H

Brain

H

(entorbinal cortex; apolipoprotein E and lipid mobilization in neuronal membrane remodeling and its relevance to Alzheimer's disease)

(hippocampus, apolipoprotein E and lipid mobilization in neuronal membrane remodeling and its relevance to Alzheimer's disease)

ANSWER 37 OF 63 CAPLUS COPYRIGHT, 2007 ACS on STN 1.1.7 A.B.

suggests Recent basic science studies show. . . the classical nuclear estrogen receptor, through which estrogen alters expression of estrogen responsive genes that play a role in apoptosis, axonal regeneration , or general trophic support. Yet another possibility is that estrogen receptors in the membrane or cytoplasm alter phosphorylation cascades estrogen in neuroprotection. Accumulated clin. evidence sthat estrogen exposure decreases the risk and delays the onset and progression of Alzheimer's disease and schizophrenia, and may

Anti-Alzheimer's agents Cognition enhancers

Schizophrenia

H

(neuroprotection by estradiol and involved mechanisms)

remediately following the lesioning, an osmotic pump to deliver serum complement and anti-galactocerebroside IgG was implanted at Tll. Subsequently, brainstem-spinal axonal rogeneration was observed in exptl. animals, as assessed by retrograde neuronal labeling with COPYRIGHT 2007 ACS on STN ANSWER 38 OF 63 CAPLUS Flurogold. L17

Alzheimer's disease

H

(complement-dependent antibody-mediated transient demyelination for promotion of neuronal regrowth and regeneration in) Parkinson's disease

1.17 AB

ANSWER 39 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN
Reactive oxygen species (ROS) are supposed to be involved in
neurodegenerative processes like Parkinson's or Alzheimer's
disease. Beside this there are an increasing number of studies indicating an
involvement of ROS in traumatic brain injury. We. . astrocytes are
able to protect retinal ganglion cells against ROS-induced oxidative
stress, (ii) astrocytes release soluble neurotrophic factors supporting RGC
axonal regeneration, and (iii) free radical production after
tissue injury may partly contribute to the failure of axonal
regeneration in the adult mammalian central nervous system.

Alsheimer's disease

H

Oxidative stress, biological Parkinson's disease

(astrocytes against neurotoxic effects of reactive oxygen species in cocultures of cortical astrocytes with regenerating postnatal retinal

11 AB

ANSWER 40 OF 63 CAPLUS COPYRIGHT 2007 ACS on STN
Aberrant GAP-43 gene expression in Alrhelmer's disease
GAP-43 is a growth-associated phosphoprotein expressed at high levels in

neurons during development, axonal regeneration, and neuritic sprouting. GAP-43 gene expression in mature neurons is probably functionally important for the structural remodeling of synapses as required for learning and establishing new memory. The widespread aberrant neuritic growth accompanied by impaired synaptic plasticity in Alzheimer's disease (AD) suggests that abnormal GAP-43 gene expression may contribute to the cascade of neurodegeneration. In the present study, end-stage.

r r

(GAP-43 gene expression in human brain in Alzheimer's disease) Neuroglia

Ħ

RL: BSU (Biological study, unclassified); BIOL (Biological study) (GAP-43 gene expression in human brain in Alzbeimer's Gene, animal

disorder Mental H

(Alzheimer's disease, GAP-43 gene expression in human brain in Alzheimer's disease)

Phospholipoproteins

RL: BOC (Biological study, unclassified); MFM (Metabolic formation); BIOL (Biological study); FORM (Formation, nonpreparative); OCCU (Courrence)

(B-50, GAP-43 gene expression in human brain in Alzbeimer's H

Nerve, disease disease) H

(degeneration, GAP-43 gene expression in human brain in Alzheimer's disease) ANSWER 41 OF 63 EMBASE COPYRIGHT (C) 2007 Elsevier B.V. All rights 117

injuries and diseases. After CNS injury, CSPGs are the major inhibitory component of the glial scar. Removal of CSPGs improves axonal regeneration and functional recovery. CSPGs may also be involved in the pathological processes in diseases such as epilepsy, stroke and Alzhaimar's disease. Several possible methods of manipulating CSPGs in the CNS have recently been identified. The development of methods to remove. reserved on STN Æ

Medical Descriptors: b

Alzheimer disease: ET, etiology epilepsy: ET, etiology eye dominance

hypothalamus hypophysis system learning memory

nerve cell lesion *nerve cell plasticity *nerve fiber regeneration

priority journal

aggrecan: EC, endogenous. stroke: ET, etiology

ANSWER 42 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights 117

evidence of developmental plasticity in these ex vivo models, demonstrating emergence of injury-stimulated neuronal progenitor cells, and neurite sprouting and axonal regenaration for and neurite sprouting and axonal regenaration as following pathway lesioning. Neuro- and axo-genesis are emerging as significant factors contributing to brain repair following many acute and reserved on STN AB

Medical Descriptors:
Alrheimer disease: ET, etiology
Parkinson disease: ET, etiology ដ

chronic.

acute disease amyotrophic lateral sclerosis: DT, drug therapy

anoxia

brain function brain injury

brain slice

cell assay

cell viability

chronic disease

*degenerative. clinical trial coculture

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reserved on STN
Reticulon (RTN) proteins are localized to the endoplasmic reticulum (ER), and are related to intracellular membrane trafficking, apoptosis, inhibiting axonal regeneration, and Alzheimer is disease. The RTN proteins are produced without an N-terminal signal peptide. Their C-terminal domain contains two long hydrophobic segments. AB

We.
Medical Descriptors:
Alzheimer disease

ដ

Golgi complex amino terminal sequence

animal cell article

carboxy terminal sequence

endoplasmic reticulum cellular distribution nydrophobicity

nerve fiber regeneration membrane transport nonhuman

nucleotide sequence

priority journal
protein determination
protein domain
protein family
protein localization

cell protein:.

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111

regeneration is therapeutically attractive for various CNS pathologies such as traumatic brain injury, stroke and Atteimer is disease. Because the RhoA pathway is involved in neurite outgrowth, tho-associated kinases (ROKS), downstream effectors of GTP-bound Rho, are Axons fail to regenerate in the adult central nervous system (CNS) following injury. Developing strategies to promote axonal reserved on STN Ā

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reserved on STN

interest. Studies focusing on animal and human olfactory bulb ensheathing cells (OECs) have heightened the expectations that OECs can enhance axonal regareration and repair demyelinating diseases. Harvest of OECs from the olfactory bulb requires highly invasive surgery, which is a major obstacle. ΑB

ANSWER 46 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
Progressive neuronal loss in Alzheimer's disease (AD) is
Progressive neuronal loss in Alzheimer's disease (AD) is
considered to be a consequence of the neurotoxic properties of
amyloid-\$\beta\$ peptides (A\beta\$). T-817MA (1-{3.12-(1.benzothiophen-5yl) ethoxyl propyl}-3-azetidinol. . . therapeutic agent for the
treatment of AD based on its neuroprotective potency against
AB-induced neurotoxicity and its effect of enhancing axonal
regeneration in the sciatic nerve axotcomy model. The
neuroprotective effect of T-817MA against A\beta\$ (1- 42) or oxidative
Stress-Induced neurotoxicity was assessed. nerve fiber regeneration nerve fiber transection central nervous system degenerative disease nerve cell necrosis brain slice *nerve injury *nerve fiber growth Alzheimer disease creatment indication nerve cell culture *neuroprotection oxidative stress *neurotoxicity disease model drug potency hippocampus orain cell nonhuman female 111 Ą ដ

testosterone actions is neuroprotection. There are some evidences supporting the hypothesis that testosterone may act protectively in neurodegenerative disorders, e.g. Alzheimer's disease (AD) mild cognitive impairment (MCI) or depression. Androgens alter also the morphology, survival and axonal regeneration of motor neurons. These hormones accelerate the regeneration of hamster facial nerve and anterior tibialis sciatic nerve in rabbits following. ANSWER 47 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN Medical Descriptors:
*central nervous system disease: DT, drug therapy
*central nervous system disease: PC, prevention *neuroprotection
Alzheimer disease: DT, drug therapy
Alzheimer disease: BT, etiology
Alzheimer disease: PC, prevention
cognitive defect: DT, drug therapy
cognitive defect: PC, prevention
depression: DT, drug therapy
depression: PC, prevention
nerve fiber regeneration 117 Ā ដ

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hormonal regulation

hamster

facial nerve sciatic nerve

nerve.

rabbit

reserved on STN Ţ

Modulation of axonal regeneration in neurodegenerative disease: Focus on Nogo.

Recent work has demonstrated that axonal regeneration in the central nervous system is limited by myelin-derived Nogo binding to an axonal Nogo Receptor. The Nogo system appears.

Tole in regulating structural plasticity. The possibility that the Nogo system contributes to pathologic and compensatory plasticity in Alzheimer's Disease is considered. Medical Descriptors: ដ ¥B

protein: EC, endogenous compound protein nogo: EC, endogenous compound myelin: EC, endogenous compound *nerve fiber regeneration
*degenerative disease: ET, etiolog
*Alzhelmer disease: ET, etiology central nervous system neuropathology nerve cell plasticity human cell animal cell article neurite human

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receptor:.

estrogen in neuroprotection. Accumulated clinical evidence auggests that estrogen exposure decreases the risk and delays the onset and progression of Alrabamar's disease and schizophrenia, and may also enhance recovery from traumatic neurological injury such as

autologous stem cell transplantation

cell isolation

donor

ex vivo study

cell lineage

Altheimer disease: DI, diagnosis Parkinson disease: DI, diagnosis neurologic disease: DI, diagnosis spinal cord injury: TH, therapy

diagnostic value

nonhuman

human

review

embryonal tissue

phenotype

animal model

priority journal

*olfactory regeneration demyelinating disease: TH, therapy olfactory epithelium nerve cell culture technique cell population mitosis H

Medical Descriptors:

stroke. Recent basic science studies show. the classical nuclear estrogen receptor, through which estrogen alters expression of estrogen responsive genes that play a role in apoptosis, acomal regeneration, or general trophic support. Yet another possibility is that estrogen receptors in the membrane or cytophasm alter phosphorylation cascades through.

*estrogen therapy *neuroprotection

ម

*schizophrenia: PC, prevention *schizophrenia: DT, drug therapy

*stroke: PC, prevention
*stroke: DT, drug therapy
*Alzheimer disease: DC, prevention
*Alzheimer disease: DT, drug therapy

nervous system development brain injury: PC, prevention brain injury: DT, drug therapy

genetic transcription

in vitro study in vivo study antioxidant activity

nonhuman

animal.

ANSWER 50 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN 111 AB

Rio blocks the neurite growth-inhibitor effects of myelin-associated glycoprotein (MAG). These findings may have clinical applications in the stimulation of axonal regeneration following injury within the CNS, and possibly in the treatment of neurodegenerative disorders.

Medical Descriptors:

ដ

structure

cell motility

degenerative disease: ET, etiology

nerve fiber regeneration Alzheimer disease: ET, etiology

human cell animal cell

short survey

*tho factor: EC, endogenous compound myelin associated glycoprotein: EC, endogenous compound rho antagonist: DV, drug development

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reserved on STN æ

Reactive oxygen species (ROS) are supposed to be involved in neurodegenerative processes like Parkinson's or Alzheimer's disease. Beside this there are an increasing number of studies indicating an involvement of ROS in traumatic brain injury. We . . . astrocytes are able to protect retinal ganglion cells against ROS-induced oxidative stress, (ii) astrocytes release soluble neurotrophic factors supporting production after tissue injury may partly contribute to the failure of axonal regeneration in the adult mammalian central

nervous system.

117

T &

ANSWER 52 OF 63 EWBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN
Aberrant GAP-43 gene expression in Alzheimer's disease.
Aberrant GAP-43 is a growth-associated phosphoprotein expressed at high levels in neurons during development, axonal regeneration, and neuritic sprouting. GAP-43 gene expression in mature neurons is probably functionally important for the structural remodeling of synapses as aberrant neuritic growth accompanied by impaired synaptic plasticity in Alzheimer's disease (AD) suggests that abnormal GAP-43 gene expression may contribute to the cascade of neurodegeneration. In the present study, end-stage.

Medical Descriptors:

ដ

*alzheimer disease article

brain cortex chromosome aberration gene expression

nemne

human tissue lewy body

priority journal receptor down regulation nerve cell plasticity nerve degeneration nerve fiber growth

white matter

ANSWER 53 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN 117

We have examined the possibility of promoting axonal regeneration within lesioned neural tissue using grafted artificial gel matrices. Polymeric matrices which feature a three-dimensional crosslinked macromolecular network were implanted. The deposition of newly synthesized extracellular molecules. This rearrangement of the brain scarring process into an organized cellular coating promoted axonal regeneration into the gels. Butragment of embryonic neurons and embryonal carcinoma (EC)-derived neurons, within the gels, was performed to explore the. ΑB

ដ

*neurosurgery *parkinson disease: SU, surgery *transplantation alzheimer disease: SU,

animal experiment controlled study animal tissue

epilepsy: SU, surgery

human tissue huntington chorea: SU, surgery korsakoff psychosis: SU, surgery

short survey

ANSWER 54 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN L17 AB

ganglia neurons was responsive to nerve growth factor (100 ng/ml). Nerve growth factor induced an increase of initial rate of axonal regeneration and influenced the survival time of these neurons. Acceptline (250 µM) did not affect the axonal regeneration but substantially attenuated the

nerve cell

*aged b

pinal ganglion alzheimer disease: ET, etiology *nerve cell culture *spinal

animal cell article

cell death

protection

survival cell

controlled study

drug effect

methodology nerve fiber degeneration

neurotropism

*levacecarnine: PD, pharm *nerve growth factor: PD, *neurotropic agent: PD,.

PD, pharmacology actor: PD, pharmacology

ANSWER 55 OF 63 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights 117 B

acquired immunodeficiency syndrome, based on regrowing or saving injured neurons. The clinical neurologist will become important in practical applications and research into prolonging neuronal survival and fostering axonal regeneration. Over the coming years, with further research, it is anticipated that patients will be treated with reserved on STN

molecular techniques underlying nerve growth are discussed.

Possible therapeutic approaches are presented for many neurologic disorders, ranging from stroke to Alzheimer's disease to these or similar modulatory.

L17

ANSWER 56 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN Progressive neuronal loss in Alsheimer's disease (AD) is considered to be a consequence of the neurotoxic properties of amyloid-beta peptides (A beta) T-817MA (1-{3·[2·(1-benzothiophen-5-yl). agent for the treatment of AD based on its neuroprotective potency against A beta-induced neurotoxicity and its effect of enhancing armal regeneration in the sciatic nerve axotomy model. The neuroprotective effect of T-817MA against A beta (1-42) or oxidative stress-induced neurotoxicity was assessed.

Organisms ST

ST ST

neuron: nervous system; central nervous system: nervous system; glial cell: nervous system; cortical neuron: nervous system Diseases

Alzheimer's disease: nervous system disease, behavioral and mental disorders

Chemicals & Biochemicals hydrogen peroxide; growth-associated protein 43; GSH; amyloid-beta:

1.17 AB

ANGWER 57 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN progressive neuronal loss in Alzheimar's disease (AD) is considered to be a consequence of the neurotoxic properties of amyloid-beta peptens (A Para). T-817MA (1-{3·[2-(1-benzothiophen-5-yl) ethoxy].

ethoxy]. agent for the treatment of AD based on its neuroprotective potency against A beta-induced neurotoxicity and its effect of enhancing account regeneration in the sciatic nerve accomy model.

The neuroprotective effect of T-817MA against A beta(1-42) or oxidative

stress-induced neurotoxicity was assessed.

Progressive neuronal loss in Alzheimer's disease (AD) is considered to be a consequence of the neurotoxic properties of amyloid- β peptides (Ab). TellYMA was screened as. therapeutic agent for the treatment of AD based on its neuroprotective potency against Ab-induced neurotoxicity and its effect of enhancing axonal regeneration in the sciatic nerve axotomy model. The neuroprotective effect of T-817MA against AB (1-42) or oxidative stress-induced neurotoxicity was assessed using. ANSWER 58 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN L17

L17 AB.

testosterone actions is neuroprotection. There are some evidences supporting the hypothesis that testosterone may act protectively in neurodegenerative disorders, e.g. Althalmer's disease (AD), mild cognitive impairment (MCI) or depression mild cognitive impairment (MCI) or depression. Androgens alter also the morphology, survival and axonal regeneration of motor neurons. These hormones accelerate the regeneration of hamster facial nerve and anterior tibialis sciatic nerve in rabbits following.

Alzheimer Disease

ដូ

Depression

Cognition Disorders

ST

SŢ

nervous system; laryngeal motor nucleus: nervous system; motor neuron: nervous system; pelvic autonomic neuron: nervous system, spinal cord Diseases

Alzheimer's disease: behavioral and mental disorders, nervous

system disease, drug therapy Alzheimer Disease (MeSH)

S SŢ

depression: behavioral and mental disorders, drug therapy Depression (MeSH) Diseases

Diseases

mild cognitive impairment; behavioral and mental.

Androgens alter also the testosterone actions is neuroprotection. There is some evidence supporting the hypothesis that testosterone may act protectively in neurodegenerative disorders, e.g. Alzhalmar's disease (AD), mild cognitive impairment (MCI), or depression. Androgens alter also the morphol., survival and axonal regeneration of motor neurons. These hormones accelerate the regeneration of hamster facial nerve and anterior tibialis sciatic nerve in rabbits following. TOXCENTER COPYRIGHT 2007 ACS on STN ANSWER 60 OF 63 L17

ANSWER 61 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN

Recent advance in adenoviral gene transfer technology for neuronal survival and axonal regeneration 117

in the treatment of Parkinson's disease, Alrholmer's disease, malignant glioma etc. is reviewed. transfer by adenovirus therapy of neuronal survival and axonal regeneration Neuron-targeted gene æ

disease. Beside this there are an increasing number of studies indicating an involvement of ROS in traumatic brain injury. We. an involvement of ROS in traumatic brain injury. We. astrocytes are able to protect retinal ganglion cells against ROS-induced oxidative ANSWER 62 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN Reactive oxygen species (ROS) are supposed to be involved in neurodegenerative processes like Parkinson's or Alzheimer's L17

stress, (ii) astrocytes release soluble neurotrophic factors supporting RGC axomal regeneration, and (iii) free radical production after tissue injury may partly contribute to the failure of axomal regeneration in the adult mammalian central

nervous system

ANSWER 63 OF 63 TOXCENTER COPYRIGHT 2007 ACS on STN
Reactive oxygen species (ROS) are supposed to be involved in
neurodegenerative processs like Parkinson's or Alzheimer's
disease. Beside this there are an increasing number of studies indicating an
involvement of ROS in tranmatic brain injury. We. . . astrocyces are
able to protect retinal ganglion cells against ROS-induced oxidative
stress, (ii) astrocytes release soluble neurotrophic factors supporting RGC
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tissue injury may partly contribute to the failure of axonal
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10537 S BATA (W) AMYLOID (W) PEPTIDE
13 NOGO (W) RECEPTOR (W) AMYAGONIST
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Toyama Chem Co Ltd, Res Labs, 2-4-1 Shimookui, Toyama 9308508, Japan Razunari hista@toyama-chemical.co.jp
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Entered STM: 21 Jun 2005 Last Updated on STM: 27 Aug 2005 Entered Medline: 26 Aug 2005

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Gross Robert E; Mei Qi, Gutekunst Claire-Anne; Torre Enrique Department of Neurosurgery, Center for Neurodegenerative Diseases, Emory University School of Medicine, Atlanta, GA 30322, USA..
Robert.gross@emoryhealthcare.org SKN85046322 (NINDS)
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Alzheimer's Disease Research Center, Neuropathology Laboratory,
Massachusetts General Hospital, Harvard Medical School, Boston, USA. ANSWER 12 OF 63 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation No. 11, pp. 1241-8. Department of Neurology, Children's Hospital, Boston, MA 02115. EY05477 (NEI) Publications ID: 27,13311 Growth factors for neuronal survival and process regeneration. Implications in the mammalian central nervous system. Abridged Index Medicus Journals; Priority Journals English Abridged Index Medicus Journals; Priority Journals Archives of neurology, (1989 Nov) Vol. 46, Journal code: 0372436. ISSN: 0003-9942. United States
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Tanhashi, Jun [Reprint Author]; Hamada, Nobuyuki; Watanabe, Hiroshi Kurume Univ, Sch Med, Dept Infect Med, Div Infect Dis, 67 Asahimachi Kurume, Pukuoka 8300011, Japan

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Williams, Melissa L., Hayes, Ronald L.; Wang, Kevin K. W. [Reprint Author]
Univ Florida, Ctr Neuroprote and Biomarkers Res, McKnight Brain Inst, POB 100256,100 S Newell Dr. Gainesville, FL 12610 USA

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Department of Neurology, and Section of Neurobiology, Yale University
School of Medicine, P.-O. Box 208018, New Haven, CT, 06510, USA
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Department of Chemistry, Boston University, Boston, MA, 02215, USA
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